most intradermal infiltrations, and the longer 27 gauge needles for infiltration along a dermal edge —for example, before lacerations are sutured— could be more widely adopted. Obviously these needles are less rigid than the 21 gauge (green) and 23 gauge (orange) needles used at present, and 21 gauge needles must be used for procedures such as deep nerve blocks and intracapsular joint injections. For giving most other local anaesthetics, however, the 27 gauge and 30 gauge needles would be adequate, and, I am sure, preferable for the patient. I am a dental surgeon, and during my current undergraduate medical training I have witnessed many patients being given unnecessarily painful injections of local anaesthetic by doctors who have received only rudimentary training in techniques. Little or no thought is being given to methods of reducing the discomfort of injection. This is at its worst in accident departments, with paediatric units giving most consideration, mainly by using topical anaesthetic creams.

Use of dental technology can facilitate Davidson and Boom's aim of providing warm sterile local anaesthetic for infiltration. Dental anaesthetics come in a variety of solutions and concentrations, with or without vasoconstrictors, and are supplied as sterilised, individually packed ampoules that fit directly into a standardised syringe, which are available in both aspirating and non-aspirating varieties. The ampoule is loaded into the syringe intact and the desired needle connected. This action punctures the ampoule so that there is no contact with the local anaesthetic before its infiltration. This system prevents the contamination of the local anaesthetic solution that is possible with repeated drawing from a stock bottle. Another bonus of using these ampoules is the ready availability of commercial thermostatic heaters, although a correctly adjusted baby's bottle warmer will do the same job at a much reduced cost. Their use also removes the need to overheat the solution to allow for cooling in the syringe, in turn reducing the dissociation of the local anaesthetic solution.

If I should need a skin laceration sutured I will visit my dentist first to have the local anaesthetic administered before attending casualty.

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1 Davidson JAH, Boom SJ. Warming lignocaine to reduce pain associated with injection. *BMJ* 1992;**305**:617-8. (12 September.)

Dermatological causes of pruritus ani

EDITOR,—We believe that there are two important omissions from D J Jones's list of dermatological conditions that can cause pruritus ani.[1]

Firstly, in female patients lichen sclerosus should be remembered. This disorder, in which characteristic white atrophic areas are found on the skin, particularly affects the genitalia but can extend to the perianal area. One of us (CIH) has observed that a fifth of women with vulval lichen sclerosus have pruritus ani, but the perianal symptoms may occur in isolation. The symptoms usually respond to potent topical steroids, but patients need to be followed up long term as there is a small risk of malignant change in the lesions.

Secondly, the importance of contact dermatitis as an aggravating factor in pruritus ani must be emphasised. In a recent study in the contact dermatitis clinic in Sheffield we patch tested 80 patients with pruritus ani. Results were positive in 55 patients. In 38 of these the reactions were to medicaments or their constituents. The commonest allergens were neomycin, fragrance mix, Peru balsam, and cinchocaine. Follow up in these patients showed an improvement or resolution of symptoms after advice in three quarters of the 55 with positive results. Patients with pruritus ani are at high risk of sensitisation from topical medicaments and toiletries, and we advise patch testing at an early stage in their management. We dispute the recommendation to use "wet wipes" as these may sensitise patients.

In conclusion, we emphasise the importance of examining the entire skin surface and the mucous membranes to obtain clues to the diagnosis and recommend that once a potent steroid has successfully alleviated symptoms the strength of steroid should be gradually reduced. In difficult cases skin biopsy may help with the diagnosis.

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1 Jones DJ. Pruritus ani. *BMJ* 1992;**305**:575-6. (5 September.)

EDITOR,—D J Jones warns about the risks of contact sensitisation in the anal region and emphasises the importance of non-irritating, hygienic measures.[1] The advice to use "moist tissues normally used for babies' bottoms" is attractive, but moist toilet paper may contain preservatives that can be allergenic. In the Netherlands a relatively new preservative, methyldibromoglutaronitrile (in Euxyl K400), is sometimes used in these products.[2]

In the past 20 months I have seen 10 patients with a positive patch test reaction to methyldibromoglutaronitrile. In seven this was related to use of moist toilet paper in the anal region. Thus before advising patients with pruritus ani to use this type of tissue paper doctors should find out what preservatives it contains.

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1 Jones DJ. Pruritus ani. *BMJ* 1992;**305**:575-7. (5 September.)
2 De Groot AC, Bruynzeel DP, Coenraads PJ, Crijns MB, Van Ginkel CJ, Van Joost T, *et al.* Frequency of allergic reactions to methyldibromoglutaronitrile (1,2-dibromo-2,4-dicyanobutane) in the Netherlands. *Contact Dermatitis* 1991;**25**:270-1.

Blood and breath alcohol concentrations

EDITOR,—Magne Nylenna and Richard Smith and subsequent correspondents discuss standardisation of units of measurement.[1 2] Measurement of alcohol concentration is a case in point.

The concentration of alcohol (ethanol) permitted in a motorist's blood is different in different countries and also within regions of the same country—for example, the United States and Australia. Besides threshold limits of blood alcohol concentration many countries also have legislation referring to the concentration of alcohol in a specimen of breath. When reporting alcohol measurements for clinical and legal purposes investigators (and academic journals) use many concentration units. This practice has caused considerable confusion.

The first statutory alcohol limits for motorists were enacted in Norway in 1936 (0·50 mg/g) and then by Sweden in 1941 (0·80 mg/g). The legal limit in Sweden was lowered to 0·50 mg/g in 1957 and then to 0·20 mg/g in 1990. Note that the concentrations of alcohol are given here in terms of mass/mass. Britain introduced its legal alcohol limit for motorists in 1967 (80 mg/100 ml), which often appears in print as the ambiguous 80 mg%. Because 1 ml of whole blood weighs on average 1·055 g,[3] 80 mg/100 ml corresponds to 76 mg/100 g.

In clinical chemistry laboratories plasma or serum is analysed more often than whole blood, and analyte concentrations are reported in accordance with the Système International d'Unitès (SI). Here the amount of substance is the mole rather than mass and the preferred unit of volume is the litre. Accordingly, a blood alcohol concentration of 100 mg/100 ml is equivalent to 21·7 mmol/l (molecular weight of ethanol=46·06). Great care is needed when alcohol determinations made in hospital clinical laboratories are cited in legal proceedings dealing with road traffic offences. Plasma and serum contain more water than whole blood and therefore also more ethanol.[4]

Threshold limits of breath alcohol concentration have been derived from existing statutory limits for blood alcohol concentration by dividing by a blood to breath conversion factor. In Britain 80 mg/100 ml divided by 2300 gives 35 µg/100 ml, which is currently the statutory limit for breath alcohol concentration. Unfortunately, different countries opted for different factors when setting their limit for breath alcohol concentration. The table gives the legal alcohol limits (blood and breath) for motorists in various countries. When handheld breath alcohol devices are used in clinical and emergency medicine the results are often reported, without qualification, as if the blood alcohol concentration had been measured directly.[5 6]

Statutory limits of alcohol in blood and breath in various countries

Country	Statutory blood alcohol	Statutory breath alcohol	Conversion (blood/breath)
Britain	80 mg/100ml	35 µg/100 ml	2300:1
Sweden	0·20 mg/g*	0·10 mg/l	2100:1
Norway	0.50 mg/g*	0·25 mg/l	2100:1
Austria	0·80 g/l	0·40 mg/l	2000:1
Holland	0·50 mg/ml	220 µg/l	2300:1
Germany	0·80 g/kg*†	‡	2100:1
France	0·80 mg/ml	0·40 mg/l	2000:1
US	0·10 g/100 ml§	0·10 g/210 l	2100:1

*1 ml of whole blood weighs 1·055 g.
†Concentrations of alcohol are determined in serum, and the result is converted into the presumed concentration in whole blood by dividing by 1·20.
‡Breath tests are used for roadside screening and not for evidential purposes.
§Applies in most US states.

The notion of reaching an international agreement about one common limit for blood or breath alcohol concentration for motorists or even the same unit of concentration (SI or otherwise) when reporting these measurements is attractive but hardly attainable.

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1 Nylenna M, Smith R. Americans retreat on SI units. *BMJ* 1992;**305**:268. (1 August.)
2 Correspondence. Americans retreat on SI units. *BMJ* 1992;**305**: 585. (5 September.)
3 Lenter C. *Geigy scientific tables.* Vol 3. 8th ed. Basle: Ciba-Geigy, 1984.
4 Jones AW, Hahn R, Stalberg HP. Distribution of ethanol and water between plasma and whole blood; inter- and intra-individual variations after administration of ethanol by intravenous infusion. *Scand J Clin Lab Invest* 1990;**50**:775-80.
5 Marks V. Measurement of breath alcohol. *Lancet* 1992;**339**:187.
6 Hahn R. Measurement of breath alcohol. *Lancet* 1992;**339**:187-8.

Osteoporosis in men

EDITOR,—We believe that David C Anderson's guidelines for investigation for clinicians in doubt about the diagnosis of osteoporosis in men require some expansion.[1] The investigations suggested fail to screen for some of the commoner causes of secondary osteoporosis in men that we and others[2] have observed (table) as well as less common but